STN Search History

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FILE 'HOME' ENTERED AT 13:59:09 ON 26 JUL 2002

=> index bioscience, pharmacology

L1    QUE (HELICOBACTER OR H) (A) PYLORI

L2    QUE (ANTIGEN OR MARKER OR ANALYTE OR PATHOGEN) (S) STOOL

L3    QUE (L1 OR L2) AND ((NUEROLOGIC (S) DISORDER) OR PDD OR PARKINSON OR DYSUT
          ONOMIC OR (PERVASIVE (A) DEVELOPMENT))

=> d rank
F1          124   USPATFULL
F2           44   INVESTEXT
F3           30   PROMT
F4           29   WPIDS
F5           29   WPINDEX
F6           19   PHIN
F7           12   BIOSIS
F8           11   MEDLINE
F9            9   IFIPAT
F10           8   EMBASE
F11           8   SCISEARCH
F12           7   DGENE
F13           5   CAPLUS
F14           5   PASCAL
F15           4   ADISALERTS
F16           4   ESBIOBASE
F17           4*  FEDRIP
F18           2   CBNB
F19           1   CABA
F20           1   DDFU
F21           1   DRUGU
F22           1   JICST-EPLUS
F23           1   TOXCENTER
F24           1   DIOGENES


=> file phin, biosis, medline, embase, scisearch, dgene


L4           65 L3
L5            48 DUP REM L4 (17 DUPLICATES REMOVED)
=> s l1
L6        90443 L1
L7            0 L1 AND ((NUEROLOGIC (S) DISORDER) OR PDD OR DYSAUTONOMIC OR
                (PERVASIVE (A) DEVELOPMENT))
L8            0 L6 AND ((NUEROLOGIC (S) DISORDER) OR PDD OR DYSAUTONOMIC OR
                (PERVASIVE (A) DEVELOPMENT))
L9         2920 PDD OR DYSAUTONOMIC OR (PERVASIVE (A) DEVELOPMENT)

L10           0 L9 AND L2
L11           0 L9 AND (PYLORI OR HELICOBACTER)
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ANSWER 12 OF 48 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 4

AN 2000:457654 BIOSIS

DN PREV200000457654

TI Parkinsonism: Differential age-trend in **Helicobacter pylori** antibody.

AU Dobbs, R. J.; Charlett, A.; Dobbs, S. M. (1); Weller, C.; Peterson, D. W.

CS (1) 2 Priory Gardens, Berkhamsted, Hertfordshire, HP4 2DR UK

SO Alimentary Pharmacology & Therapeutics, (September, 2000) Vol. 14, No. 9, pp. 1199-1205. print.
ISSN: 0269-2813.

DT Article

LA English

SL English

AB Background: Parkinsonism is associated with prodromal peptic ulceration. Dopamine antagonists provoke experimental ulcer, dopaminergic agents protect, and might inhibit growth of **Helicobacter pylori**. Objective: To describe the relationship between **H. pylori** serology and parkinsonism. Methods: Serum **H. pylori** anti-urease-IgG antibody was measured in 105 people with (idiopathic) parkinsonism, 210 without, from same locality. None had received specific eradication therapy. Results: Controls showed a birth-cohort effect: antibody titre rose from 30 to 90 years ($P < 0.001$). Parkinsonism obliterated this (disease status cntdot age interaction, $P < 0.05$), the differential age trend not being attributable to social class. Those with diagnosed parkinsonism were more likely to be seropositive (odds ratio 2.04 (95% CI: 1.04, 4.22) $P < 0.04$) before 72.5 years. Overall, titre fell ($P = 0.01$) by 5 (1, 9)% per unit increase in a global, 30-point rating (median 14 (interquartile range 10.5, 17)) of disease severity. No individual category of anti-parkinsonian medication (92% taking) had a differential lowering effect. Conclusions: Higher prevalence of seropositivity in parkinsonism, before 8th decade, may be due to host susceptibility/reaction, or, conversely, infection with particular **H. pylori** strain(s) lowering dopaminergic status. Absence of a birth cohort effect in parkinsonism, despite similar social class representation, may be consequent on eradication, spontaneous (gastric atrophy) or by anti-parkinsonian medication.

L5 ANSWER 13 OF 48 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 2001:73545 BIOSIS

DN PREV200100073545

TI Insights into the natural history of idiopathic Parkinsonism in relation to **Helicobacter pylori** anti-urease antibody titre.

AU Dobbs, S. M. (1); Charlett, A.; Dobbs, R. J. (1); Weller, C. (1)

CS (1) Therapeutics in the Elderly, Northwick Park and St Mark's Hospital, Harrow, HA1 3UJ UK

SO British Journal of Clinical Pharmacology, (October, 2000) Vol. 50, No. 4, pp. 389. print.
Meeting Info.: British Pharmacological Society, Clinical Pharmacology Section Cardiff, Wales, UK July 12-14, 2000 British Pharmacological Society
. ISSN: 0306-5251.

DT Conference

LA English

SL English

L5 ANSWER 14 OF 48 MEDLINE DUPLICATE 5

AN 2001195684 MEDLINE

DN 21129183 PubMed ID: 11233523

TI Evidence based medicine and extradigestive manifestations of

Helicobacter pylori.
AU De Koster E; De Bruyne I; Langlet P; Deltenre M
CS Department of Gastroenterology, CHU Brugmann UVC (VUB-ULB), Brussels, Belgium.
SO ACTA GASTROENTEROLOGICA BELGICA, (2000 Oct-Dec) 63 (4) 388-92. Ref: 27
Journal code: 0414075. ISSN: 0001-5644.
CY Belgium
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200104
ED Entered STN: 20010410
Last Updated on STN: 20010410
Entered Medline: 20010405
AB A putative pathogenetic role has been ascribed to **Helicobacter pylori** in several extradigestive diseases, including vascular (atherosclerosis and ischaemic heart disease, primary Raynaud phenomenon, primary headache), autoimmune (Sjogren's syndrome, Henoch-Schonlein purpura, autoimmune thyroiditis, idiopathic arrythmias, **Parkinson**'s disease, nonarterial anterior optic ischemic neuropathy), and skin diseases (chronic idiopathic urticaria, rosacea, alopecia areata), sideropenic anemia, growth retardation, late menarche, extragastric MALT lymphoma, diabetes mellitus, hepatic encephalopathy, sudden infant death syndrome, and anorexia of aging. We examine critically the strength of the evidence linking these diseases to **Helicobacter pylori**, using ischaemic heart disease as an example of epidemiological techniques, and skin diseases as an example of treatment studies. By the standards of evidence-based medicine, studies have been often of low quality. The best evidence usually is not indicative of a role for **Helicobacter pylori** in these diseases.

L5 ANSWER 15 OF 48 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:176696 BIOSIS
DN PREV200100176696
TI Systemic cortisol response to **Helicobacter pylori** vacuolating toxin in idiopathic parkinsonism and controls.
AU Charlett, A.; Weller, C. (1); Oxlade, N. (1); Peterson, D. W. (1); Dobbs, S. M. (1); Dobbs, R. J. (1)
CS (1) Therapeutics in the Elderly, Northwick Park and St Mark's Hospital, Harrow, HA1 3UJ UK
SO British Journal of Pharmacology, (December, 2000) Vol. 131, No. Proceedings Supplement December, pp. 220P. print.
Meeting Info.: Meeting of the British Pharmacological Society Bradford, England, UK September 06-08, 2000 British Pharmacological Society
. ISSN: 0007-1188.
DT Conference
LA English
SL English

L5 ANSWER 16 OF 48 MEDLINE
AN 2000497310 MEDLINE
DN 20366366 PubMed ID: 10904422
TI Link between **Helicobacter pylori** infection and idiopathic parkinsonism.
AU Dobbs S M; Dobbs R J; Weller C; Charlett A
CS Therapeutics in the Elderly, Research Group, Northwick Park & St Mark's Hospitals, Harrow, UK.. dobbs@wellers.demon.co.uk
SO MEDICAL HYPOTHESES, (2000 Aug) 55 (2) 93-8.

Journal code: 7505668. ISSN: 0306-9877.
CY SCOTLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200010
ED Entered STN: 20001027
Last Updated on STN: 20001027
Entered Medline: 20001013
AB The conventional concept for an environmental cause of idiopathic parkinsonism is an insult (e.g. neurotoxin or encephalitis), superimposed on age-related attrition of nigral dopaminergic neurons, and temporally remote from neurological diagnosis. To the contrary, we describe the fit of **Helicobacter pylori**. This commonest of known bacterial infections, usually acquired in childhood, persists, and has been linked with peptic ulcer/non-ulcer dyspepsia, immunosuppression and autoimmunity. Acquired immunosuppression, predisposing to auto-immunity, is assessed as a model for the pathogenesis of parkinsonism and parkinsonian-like attributes of ageing. Eradication of a trigger has potential to change the approach to parkinsonism, just as it did to peptic ulcer. The tenet of inevitable age-related attrition of dopaminergic neurons may also require revision.

WEST Search History

DATE: Friday, July 26, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L6	l3 and (pylori and (detect with (disease disorder)))	1	L6
L5	l3 and (pylori or (detect with (disease disorder)))	13	L5
L4	5039607.pn.	1	L4
L3	(fecal stool) with (assay test immunoassay) same antigen and pathogen	33	L3
L2	5198365	10	L2
DB=PGPB; PLUR=YES; OP=OR			
L1	fallon.in. and pylori.clm.	1	L1

END OF SEARCH HISTORY

WEST Search History

DATE: Friday, July 26, 2002

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR		
L30	L29 and l14	4	L30
L29	L28 and @ad<20001116	91	L29
L28	L19 and (neurolog$5 Parkinson pdd (pervasive adj development) dysautonomic)	111	L28
L27	L25 and pylori	1	L27
L26	L25 and l14	0	L26
L25	L24 and (test detect assay) same bacteria	27	L25
L24	L23 and @ad<20001116	70	L24
L23	L19 and stool same immunoassay	76	L23
L22	L21 and (neurolog$5 Parkinson pdd (pervasive adj development) dysautonomic)	4	L22
L21	L20 and @ad<20001116	90	L21
L20	L19 and l14	114	L20
L19	stool and antigen and (anal$5 or assay or detect)	1269	L19
L18	L17 and Pylori same Parkinson	60	L18
L17	L14 and (Parkinson)	137	L17
L16	L14 and (pdd or (pervasive adj development))	2	L16
L15	L14 and (dysautonomic)	1	L15
L14	L5 or H adj pylori	3053	L14
L13	L12 and (detect stool marker) same pylori	4	L13
L12	L5 and neurologic$2	101	L12
L11	L9 and Parkinson same pylori	43	L11
L10	L9 and stool	8	L10
L9	L5 and (Parkinson)	119	L9
L8	L5 and (pdd or (pervasive adj development))	2	L8
L7	L5 and (dysautonomic)	1	L7
L6	L5 and (pdd or (pervasive adj developement))	2	L6
L5	Helicobacter same pylori	2797	L5
L4	Heliobacter same pylori	103	L4
L3	L2 and (marker antigen) same stool	20	L3
L2	(Helicobacter adj pylori) and stool	188	L2
	DB=USPT; PLUR=YES; OP=OR		

L1	(Helicobacter adj pylori) and stool	144	L1

END OF SEARCH HISTORY

Results are shown in relevance ranked order. **To re-phrase your existing search criteria**, press the 'Back' button on your browser. [Help with Searching]

Search Results

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Clear | Get All Checked Abstract(s)

JECH

J. Epidemiol. Community Health 55: 1a-56a. [Full Text] [PDF] [Publisher's Correction]
Society for Social Medicine and the International Epidemiological Association European Group
Abstracts of oral presentations
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SO British Journal of Clinical Pharmacology, (October, 2000) Vol. 50, No. 4, pp. 389. print.
Meeting Info.: British Pharmacological Society, Clinical Pharmacology Section Cardiff, Wales, UK July 12-14, 2000 British Pharmacological Society
. ISSN: 0306-5251.

TI Evidence based medicine and extradigestive manifestations of Helicobacter pylori.
AU De Koster E; De Bruyne I; Langlet P; Deltenre M
CS Department of Gastroenterology, CHU Brugmann UVC (VUB-ULB), Brussels, Belgium.
SO ACTA GASTROENTEROLOGICA BELGICA, (2000 Oct-Dec) 63 (4) 388-92. Ref: 27
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appears to hold true even at very low levels of dextropropoxyphene. The ethanol effect in this series, however, accounted for only 4% of the total variance present.

1 Finkle BS, *et al. J Forensic Sci* 1976; **21**: 706.
2 Carson DJL, *et al. Lancet* 1977; i: 894.
3 Ali NA, *et al. Br J Clin Pharmacol* 1985; **20**: 631.

This analysis confirms the reported interaction between ethanol and dextropropoxyphene in fatal overdoses and gives an indication of the strength and nature of this relationship.

4 Girre C, *et al. Eur J Clin Pharmacol* 1991; **41**: 147.
5 Parfitt K (Ed). *Martindale*, 32nd Edition. Pharmaceutical Press.

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Insights into the natural history of idiopathic Parkinsonism in relation to *Helicobacter pylori* anti-urease antibody titre

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Idiopathic parkinsonism may be an extra-gastric manifestation of *Helicobacter pylori* infection [1]. In healthy subjects, serum anti-urease antibody titre rose with age. This, expected, birth cohort effect was absent in parkinsonism, [2]. The odds for seropositivity, in sufferers, was twice that in controls, below 72.5 years, less than unity thereafter. The differential age trend suggests more aggressive infection, perhaps with particular *H. pylori* strain(s), and/or more flamboyant host reaction, in parkinsonism. Greater, consequent, gastric atrophy might result in greater lightening of microbial load in parkinsonism. Immunoblot antibody profiling supports strain difference [3].

We explore the relationship of established global ratings and time since diagnosis of idiopathic parkinsonism to *H. pylori* antibody titre, in 105 sufferers (55 men, 50 women; median (interquartile range) age 74 (62 to 78) years). Disease severity was measured by the Webster (30 point) rating, functional impairment by the Hoehn & Yahr (I–V) rating. Median value for severity was 14 (interquartile range 10.5 to 17; range 4 to 25), that for functional disability III (range II (32%) to IV (33%)). None had been treated for *H. pylori* infection. Enzyme-linked immunosorbent assay measured IgG antibody against a known fraction of *H. pylori* urease (SIA *Helicobacter pylori* (HM-CAP), Sigma-Aldrich Ltd, Poole). A calibration curve converts absorbence to an 'ELISA value' (EV). The between assay coefficients of variation, for samples assayed in duplicate, were 13.0, 8.0 and 6.0%, at EVs of 0.8, 2.4 and 5.9. A generalized linear model was fitted to assess associations between the dependent variable, EV, and candidate covariates. A gamma probability distribution was assumed for EV, a log link being used to relate the candidates (global ratings and time since diagnosis) and the known covariate (gender, but not age [2]).

H. pylori antibody titre appeared to have a large effect on disease severity rating. EV fell by 5.4 (95% C.I. 1.2, 9.3) % per unit rise in the rating ($P=0.01$). Splitting the rating into four categories (<10, 10 to <15, 15 to <20, ≥ 20), to embrace any non-linearity, did not improve the fit (likelihood ratio test, $\chi^2=1.84$, DF=2, $P=0.4$). EV was higher (63 (6, 151) %, $P=0.03$) in mild/moderate functional impairment (stage III) than in minimal (II), but similar (-8 (-42, 46) %, $P=0.7$) in severe (IV) disability to in minimal. Neither time from diagnosis (median (interquartile range) 70 (32–120) months), nor the time for which the condition had been judged sufficiently severe to require levodopa (48 (10, 96) months), contributed to prediction of EV. The two global ratings did show some congruity: 47% (adjusted r^2) of the variance in severity can be explained by functional impairment. There was no significant relationship between time from diagnosis and severity rating. Time from diagnosis was not different in stage III, or IV, to that in stage II. The global scores were measuring features of the disease that were complementary to duration from the threshold for diagnosis or levodopa prescription.

The findings are compatible with greater destruction of the environment, in which *H. pylori* thrives, in more severe parkinsonism and as the functional impairment progresses from mild/moderate to severe. They may explain the lack of birth cohort effect on *H. pylori* titre in parkinsonism. Moreover, the implication is that, if the organism drives an immune/inflammatory process [4] resulting in damage to the basal ganglia, then that process may spontaneously abort, or be terminated therapeutically. Parkinsonism, presenting in older-age, is often relatively quiescent: minimal functional impairment might be the consequence of less virulent strain(s).

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